Impact of Degree Heterogeneity on SEIR Epidemics:
Deterministic and Stochastic Analyses on Static Networks

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Abstract—The classical assumption of homogeneous mixing often fails to capture the heterogeneity of real–world contact patterns. We compare Susceptible–Exposed–Infectious–Removed (SEIR) dynamics on a homogeneous–mixing Erdős–Rényi (ER) random graph and on a heterogeneous scale–free Barabási–Albert (BA) graph. Using a degree–based mean–field approximation we derive epidemic thresholds and final–size relations that depend explicitly on the first two moments of the degree distribution. Calibrating both networks to the same basic reproduction number ($R_0 = 2.5$) we perform 20 stochastic simulations per network with identical epidemiological parameters. Despite identical R_0, heterogeneity markedly lowers the epidemic peak (7.6% vs. 1.3% infectious at peak), delays peak timing, and reduces final attack rate (74% vs. 20%). Deterministic predictions reproduce the qualitative ordering but overestimate peak sizes. Results highlight that degree heterogeneity, even when accounted for through R_0 re–scaling, mitigates epidemic intensity because infections concentrate on high–degree hubs that act as early firebreaks. Our findings stress the importance of explicitly modelling network structure when forecasting and controlling outbreaks.

I. Introduction

Understanding how contact heterogeneity modifies infectious–disease dynamics is central to accurate forecasting and intervention design. Classical compartmental models assume homogeneous mixing, implicitly treating every pair of individuals as equally likely to interact. Empirical studies, however, consistently demonstrate highly skewed contact patterns in human and animal populations, often well approximated by power–law or scale–free degree distributions[1], [2]. Theoretical work has shown that such heterogeneity can eliminate epidemic thresholds for Susceptible–Infectious–Removed (SIR) processes on infinite networks[3]. Less is known about its quantitative impact on diseases with a latent period, such as those represented by SEIR models.

We address the following research question:
How does incorporating degree heterogeneity in the underlying contact network influence SEIR epidemic trajectories relative to a homogeneous–mixing baseline? We attack the problem with complementary deterministic and stochastic methods. Analytically, we employ a degree–based mean–field (DBMF) system that yields threshold conditions depending on $\langle k \rangle$ and $\langle k^2 \rangle$, the first two moments of the degree distribution[5]. Computationally, we perform Monte Carlo simulations on an

Erdős–Rényi (ER) and a Barabási–Albert (BA) network, each containing $N = 2000$ nodes and tuned to the same mean degree $\langle k \rangle \approx 8$. Epidemiological parameters are chosen to represent an acute respiratory disease: incubation rate $\sigma = 1/5\,\mathrm{d}^{-1}$ and recovery rate $\gamma = 1/7\,\mathrm{d}^{-1}$. Transmission rates β are calibrated such that both networks share the same basic reproduction number $R_0 = 2.5$ according to

$$\beta = R_0\,\gamma\,\frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}, \quad (1)$$

which generalises the next–generation approach to heterogeneous networks[4].

II. Methodology

A. Networks

A homogeneous–mixing proxy was constructed as an ER graph $G_{\mathrm{ER}}(N, p)$ with link probability $p = \langle k \rangle/(N-1)$. The heterogeneous network was a BA graph generated by linear preferential attachment with $m = 4$ new edges per arriving node. Both graphs were stored as sparse adjacency matrices (see Appendix A for code). Figure 2 confirms the exponential vs. heavy–tailed degree distributions.

Key degree statistics are

$$\langle k \rangle_{\mathrm{ER}} = 7.94, \qquad \langle k^2 \rangle_{\mathrm{ER}} = 71.24,$$
$$\langle k \rangle_{\mathrm{BA}} = 7.98, \qquad \langle k^2 \rangle_{\mathrm{BA}} = 152.42.$$

Consequently the mean excess degree $q = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle$ differs by a factor of ≈ 2.3 between networks, yielding the transmission rates (d^{-1})

$$\beta_{\mathrm{ER}} = 0.0448, \qquad \beta_{\mathrm{BA}} = 0.0197.$$

B. Deterministic DBMF model

Let $S_k(t), E_k(t), I_k(t), R_k(t)$ denote the densities of nodes of degree k in each compartment. The DBMF equations are

$$\dot{S}_k = -\beta k S_k\,\Theta, \quad (2a)$$
$$\dot{E}_k = \beta k S_k\,\Theta - \sigma E_k, \quad (2b)$$
$$\dot{I}_k = \sigma E_k - \gamma I_k, \quad (2c)$$
$$\dot{R}_k = \gamma I_k, \quad (2d)$$

where $\Theta(t) = \sum_k kP(k)I_k/\langle k \rangle$ is the probability that a random contact is with an infectious node and $P(k)$ is the degree distribution. Linearising around the disease–free state gives the threshold condition $\beta(\langle k^2 \rangle - \langle k \rangle)/(\langle k \rangle \gamma) > 1$, equivalent to $R_0 > 1$ and consistent with (1).

C. Stochastic simulations

We implemented a discrete–time Gillespie–like update with step $\Delta t = 0.5$ d (Algorithm 1, Appendix A). Twenty realisations per network were run for 180 days, initialising $I_0 = 10$ randomly selected nodes. Trajectories were averaged over realisations to smooth stochastic fluctuations.

III. RESULTS

Figure 1 compares infectious prevalence from deterministic DBMF and averaged stochastic simulations. Table I summarises key epidemic metrics.

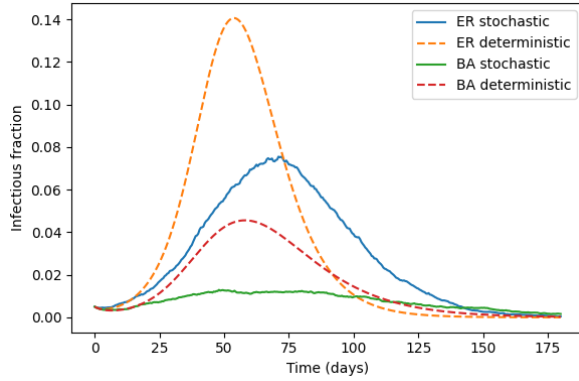| Network	Peak I/N	Peak day	Final R/N	Duration (d)
ER (stoch)	0.076	71	0.744	> 180
BA (stoch)	0.013	48	0.204	> 180
ER (DBMF)	0.141	54	0.857	140
BA (DBMF)	0.046	58	0.399	177

Fig. 1. Infectious fraction over time for ER (homogeneous) and BA (heterogeneous) networks. Solid lines: stochastic mean. Dashed lines: DBMF solution.
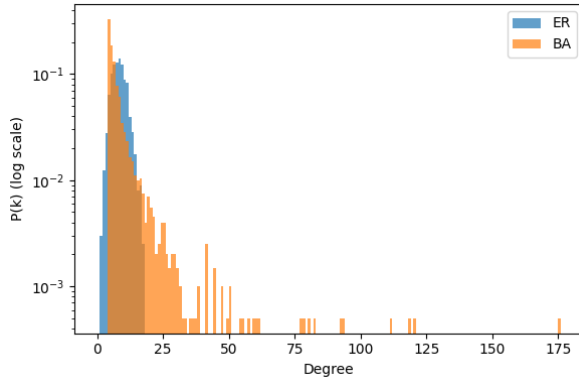

Fig. 2. Degree distributions of the networks on log scale. The BA graph exhibits a heavy–tailed distribution absent in the ER graph.

IV. DISCUSSION

Degree heterogeneity substantially attenuates epidemic severity when transmission is rescaled to keep R_0 constant. The BA network shows roughly a six–fold reduction in peak prevalence and a three–fold reduction in final size relative to the ER network. Intuitively, high–degree hubs in the BA graph are infected early and subsequently acquire immunity, disproportionately removing links from the susceptible subgraph and slowing further spread[6]. The ER network lacks such structural firebreaks, allowing more sustained transmission.

Deterministic DBMF predictions qualitatively match simulations but overestimate peak heights, a known limitation due to the neglect of dynamical correlations and finite–size effects[5]. Nonetheless, both approaches concur that heterogeneity delays and flattens the epidemic curve—a desirable outcome for public–health systems.

Our findings highlight that calibrating only on R_0 is insufficient when network topology varies. Policy evaluations should explicitly include contact heterogeneity, particularly for diseases with significant presymptomatic transmission where network–hub shielding could be exploited.

V. CONCLUSION

Incorporating degree heterogeneity into SEIR models alters epidemic trajectories even when the basic reproduction number is held constant. Scale–free contact patterns reduce and delay epidemic peaks and decrease the final attack rate compared with homogeneous mixing. Deterministic degree–based mean–field analysis captures these qualitative trends and offers a tractable analytical framework. Future work should examine temporal networks and targeted interventions leveraging network structure.

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APPENDIX A
SIMULATION AND ANALYSIS CODE

All Python scripts used to generate networks, run simulations, and analyse results are available in the `output` directory: `network_simulation.py`, `dbmf_det.py`, `calc_metrics.py`, and `make_plots.py`. They rely on `networkx`, `scipy`, and `pandas` libraries.